Understanding the impact of alcohol on blood pressure and hypertension: From moderate to excessive drinking

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ABSTRACT

Observational studies report a strong positive linear association between alcohol consumption and blood pressure but also suggested a lower cardiovascular risk with light drinking compared with abstainers. However, this potential cardioprotective effect of low-to-moderate alcohol intake seems attributable to a healthier life style in these individuals. Hence, more recent epidemiological and genetic Mendelian randomization studies indicated a continuous nonlinear positive relationship between alcohol intake and blood pressure (BP). The risk for hypertension increases in both men and women, if daily alcohol intake is at least one to two drinks (about 10–20 g alcohol) per day. Alcohol reduction close to abstinence is associated with a 3.3 and 2.0 mm Hg reduction in systolic BP and diastolic BP. A dose-dependent relationship between alcohol intake and BP was observed particularly in heavy drinkers consuming at least 6 drinks/day. In this group, more profound BP reductions can be expected by reducing alcohol intake. Additionally, both trial data and observational literature support the hypertensiogenic effect of binge drinking, which together with uncontrolled hypertension, is the most important risk factor for intracranial hemorrhage. Consequently, excessive (binge) drinking should be avoided, and patients with high risk for intracranial bleedings should be advised accordingly. Recommendations in different guidelines vary regarding the upper limits and definition of drinks, and recommendations for sex-specific upper limits for alcohol intake appear questionable. Moderation in alcohol intake and implementation of alcohol-free days during the week in both men and women who consume drinks that contain alcohol are recommended to improve BP control and overall health.

Key words: alcohol**,** blood pressure, cardiovascular risk, hypertension, stroke

INTRODUCTION

In 2019, global alcohol consumption averaged 5.5 liters of pure alcohol per person, with significant variations observed worldwide [1]. The range varies from region to region, with the lowest consumption, such as the Middle East at just 0.3 liters, to the highest consumption, notably in Europe at 9.2 liters [1]. Specifically in Europe, men consume an average of 14.9 liters *per capita*, while women consume 4.0 liters [1]. Alcohol attributable mortality is considerably higher among men, accounting for approximately 2.07 million fatalities, as opposed to 374 000 among women [2]. In both sexes, high systolic blood pressure (SBP) is a leading risk factor for deaths, responsible for 10.8 million fatalities in 2019 [2]. Alcohol consumption contributes to disability, measured in Disability-Adjusted Life Years, representing 6.3% of the global burden of disease. Following closely, high SBP accounts for 6% of Disability-Adjusted Life Years, particularly in the younger age group (25–49 years) [2].

What is the accepted definition and terminology for alcohol intake, particularly in the context of the "drink concept"?

A standard drink is a measurement of alcohol consumption, serving as a benchmark for the amount of pure alcohol in a beverage [3]. The term drink was introduced for pragmatic reasons, because this construct is used in epidemiological studies on alcohol consump-

Table 1. Recommendations of scientific societies about alcohol consumption in hypertension management guidelines

Guidelines	Recommendation(s)
ESC/ESH (2018) [8] ESH 2023 [4]	Less than 14 g for men and 8 g for women per week, alcohol free days, avoid binge drinking Below 3 drinks/day and close to abstinence in both men and women, avoid binge drinking
ACC/AHA (2017) [9]	Less than 2 standard drinks daily for men and 1 standard drink for women
ISH (2020) [10]	2 standard drinks for men and 1.5 for women, avoid binge drinking
Polish Society of Hypertension (2019) [11]	Up to 20–30 g of pure alcohol in men per day, but not more than 140 g per week and up to 10–20 g of pure alcohol per day in women but not more than 80 g per week, alcohol free days, avoid binge drinking
The Chinese Society of Hypertension (2018) [12]	Up to 25 g for men and 15 g for women per day and up to 140 g for men and 80 g for women per week
The Korean Society of Hypertension (2018) [13]	Less than 2 drinks per day for men, less than 1 for women. Appropriate moderate daily amount of alcohol is less than 20–30 g for men; 10–20 g for women. People with lower-than average body weight are permitted half of the recommended amount
The Japanese Society of Hypertension (2019) [14]	Up to 20–30 ml ethanol/day (man) or up to 10–20 ml ethanol/day (woman)

1 standard drink (US) =14 g pure alcohol; 1 standard drink (ISH) = 10 g alcohol; drink (Korean) not clear

Abbreviations: ACC, American College of Cardiology; AHA, American Heart Association; ESC, European Society of Cardiology; ESH, European Society of Hypertension

tion based on self-reported alcohol intake as defined by drinks per day [4]. However, with no global consensus on the quantity of alcohol contained in a standard drink, definitions vary across countries and regions. In Europe, in general one drink contains between 10–12 g of alcohol [3]. In the United Kingdom, "units" of alcohol are used with one unit equating to about 8 g of alcohol [5]. The number of units in a drink depends on its size and alcohol strength by volume. In the United States, a standard drink contains roughly 14 g of pure alcohol. The World Health Organization (WHO) offers another standard, defining one unit as equivalent to 10 g of pure ethanol [6]. For example, one unit represents 125 ml of wine, 250 ml of beer, or 40 ml of vodka. This definition has been accepted by more countries than any other [5]. The inconsistency and variability in the definition of a standard drink create challenges in public health communication, research, guidelines, and accurate assessment of alcohol-related risks and effects.

Alcohol consumption is a common modifiable risk factor for a range of diseases, including cardiovascular (CV) conditions [4, 7]. National and international scientific societies issue guidelines and recommendations for acceptable alcohol consumption, typically classifying different levels of risk based on the amount of pure alcohol in grams or standard drinks, as seen in guidelines for hypertension management. Nevertheless, these recommendations frequently present divergent and incongruent definitions for acceptable alcohol intake (Table 1). The consensus on acceptable alcohol consumption for individuals with hypertension varies, but generally, moderation is the key. Moderate drinking is often defined by scientific societies as up to one drink per day for women and up to two drinks per day for men; therefore, placing a higher threshold for moderate drinking for men than women. This notion appears peculiar considering that the number of alcohol-related deaths in men is more than four times greater than in women [2]. Differences in blood pressure (BP) sensitivity to alcohol between sexes might explain these sex-specific recommendations. Women have a reduced ability to metabolize alcohol by first-pass metabolism and gastric mucosal alcohol dehydrogenases [15]. Furthermore, variations in body fat distribution, body size, and drinking habits contribute to these differences, as men tend to consume alcohol more frequently and in larger amounts compared to women [16].

How does drinking alcohol impact BP?

Numerous of both acute and chronic conditions, have proposed various mechanisms related to neural and hormonal reactions. These mechanisms involve sympathetic nervous system activation, increased renin and cortisol levels, modification of carotid baroreceptor response, and increased peripheral vascular muscle tone [17]. Additionally, experimental evidence indicates that alcohol consumption affects addversely endothelial function, reducing nitric oxide production and promoting generation of reactive oxygen species and oxidative stress [18] (Figure 1).

Acute effects of alcohol intake on BP

Acute alcohol consumption has a biphasic effect on BP with an initial dose-dependent reduction in both SBP and diastolic BP (DBP) and an increased heart rate (up to 17 hours after exposure). This is due to acute vasodilatory effects followed by a later rebound in BP [19]. A Cochrane meta-analysis [20] of 32 randomized controlled trials involving 767 participants found no effect of low-to-moderate alcohol intake (14 to 28 g) on BP when compared to placebo. However, high-dose alcohol (>30 g) resulted in an initial reduction in SBP (–3.5 mm Hg) and DBP (–1.9 mm Hg) within 6 hours followed by increased SBP (+3.7 mm Hg) and DBP (+2.4 mm Hg) after more than 13 hours.

While the relationship between the immediate effects of alcohol and the long-term elevation of BP remains unclear, there is substantial risk associated with heavy consumption, often referred to as binge drinking.

How hazardous is binge drinking?

Heavy episodic drinking, known as binge drinking, is a pattern of alcohol intake that may be defined as consuming 5 drinks or more in a row for men or ≥4 drinks for women

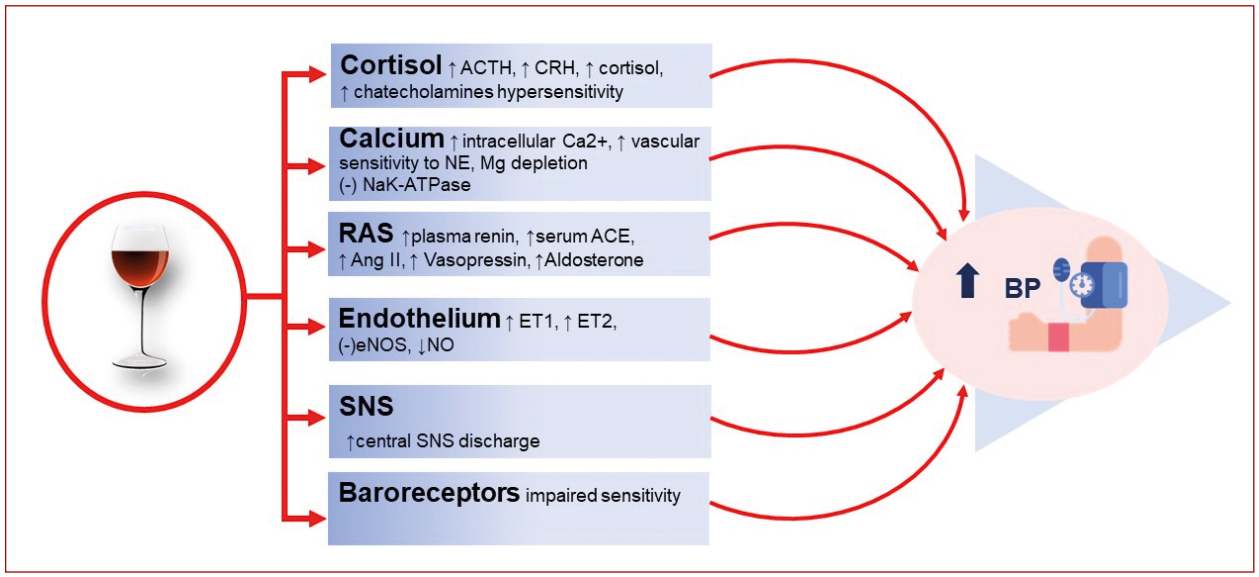

Figure 1. Mechanisms mediating the adverse effects of alcohol on blood pressure

Abbreviations: ACE, angiotensin-converting enzyme; ACTH, adrenocorticotropic hormone; Ang II, angiotensin II; CRH, corticotropin-releasing hormone; eNOS, endothelial nitric oxide synthase; ET, endothelin; NE, norepinephrine; NO, nitric oxide; SNS, sympathetic nervous system

per occasion within the past 30 days [21]. Frequent binge drinking over an extended period is associated with increased BP levels in adolescence and adulthood and may be implicated in the development of hypertension [22–24]. A large population-based study [25] reported a significant, positive association with both infrequent binge drinking (less than once a week) (OR, 1.23; 95% CI, 1.02–1.49) and frequent binge drinking (heavy consumption) (OR, 1.64; 95% CI, 1.22–2.22) with hypertension in adolescence. The highest risk was observed when frequent binge drinking was exercised in both adolescence and early adulthood (OR, 2.43; 95% CI, 1.13–5.20) [25].

In addition to its role in hypertension development, binge drinking is also linked to increased risk of stroke and is considered an independent risk factor for mortality from ischemic heart disease [26]. Heavy alcohol consumption increases the risk of both intracerebral hemorrhages (ICH) and subarachnoid hemorrhages mainly due to alcohol-induced hypertension and impaired hemostasis [27]. A vasoconstriction caused by episodic alcohol intoxication increases both SBP and DBP during intoxication and when blood alcohol levels are decreasing, usually at night, both pressure levels fall below the basic level [28]. This fluctuation in BP may contribute to the rupture of small cerebral arteries with blood entry into the brain parenchyma triggering hypertensive ICH [29]. Furthermore, vascular endothelium dysfunction promotes the occurrence of incident cerebral microbleeds [30]. ICH accounts for 20%–30% of all acute strokes, with notably high mortality rates exceeding 50% and a range of dismal functional and cognitive sequelae [31, 32]. Hypertension and excessive alcohol consumption are major risk factors for this condition [32]. Moreover, binge drinking further amplifies the mortality risk attributable to ICH for individuals with chronic drinking habits [33].

Chronic effects of alcohol on BP/incidence of hypertension: Threshold and sex-specific differences

The association between alcohol consumption and hypertension was first reported by Lian back in 1915 [34] and has been ever since documented by numerous studies [35]. A community study on atherosclerotic risks [35] examined the longitudinal association between patterns and amounts of alcohol consumption and the incidence of hypertension in a large multiethnic cohort. The study revealed a higher risk of hypertension in white men drinking ≥210 g of alcohol per week but no evident risk at low-to-moderate consumption levels (1–209 g per week) compared to non-drinkers [35].

A meta-analysis by Roerecke et al. [36] incorporated data from 20 cohort studies (125 907 men and 235 347 women, with 90 160 reported cases of incident hypertension) revealing sex-related differences in the association and a dose-response relationship. In men, a small (relative risk [RR] 1.19; 95% CI, 1.07–1.31) but significant risk of hypertension started already at a consumption level of 1–2 drinks (12–24 g of pure alcohol) per day. This risk increased significantly and consistently with higher alcohol intake: RR, 1.51 (0.95% CI, 1.30–1.76) for 3–4 drinks, and RR, 1.74 (0.95% CI, 1.35–2.24) for ≥5 standard drinks daily. In contrast, in women, an increased risk was observed for consumption of ≥3 drinks per day (RR, 1.42; 95% CI, 1.22–1.66) [36]. Hence, an increased risk could be observed with the lowest daily ingested amount in men but in women, the risk was only evident when the intake was beyond 2 drinks per day. Notwithstanding, women exhibited a steeper dose-response curve and higher effect size than men at the corresponding drinking levels [36].

The association between alcohol intake and incident hypertension at low levels of consumption was further scrutinized by another meta-analysis of 31 cohort studies (414 477 participants) by Liu et al. [37]. At 10 g/d, men showed a significantly higher risk (RR, 1.14; 95% CI, 1.07, 1.20) than women (RR, 0.98; 95% CI, 0.89, 1.06), with a comparable effect size as reported by Roerecke et al. [36]. In the dose-response analysis of both men and women, each 10 g/d increment of consumption increased the RR of hypertension by 6% (RR, 1.06; 95% CI, 1.05, 1.08) compared with non-drinkers while a consumption of 50 g/d, increased the risk by 35%. The study highlighted the sex-specific difference in the dose-response association evident also at low-level of alcohol consumption [37].

Recently, a dose-response meta-analysis of nonexperimental cohort studies indicated a linear relationship between alcohol intake and BP with no evidence of a threshold for the association [38]. The meta-analysis included 19 548 participants with a median follow-up of 5.3 years using a dose-response 1-stage meta-analytic approach; hence, the authors were able to assess a much broader range of exposure including low levels. Alcohol intake at 12 g of alcohol per day was associated with a small increase in SBP of 1.25 mm Hg (95% CI, 0.49–2.01) compared with nondrinkers [38]. Statistically significant differences in both SBP and DBP were more pronounced at higher levels such as 24 g/day where SBP increased by 2.48 mm Hg (95% CI, 1.40–3.56) and DBP by 2.03 mm Hg (95% CI, 1.19–2.86). Sex-specific analyses showed an almost linear association between baseline alcohol intake and SBP changes in both men and women, with no threshold for the association in either sex [38]. The association had a steeper slope for SBP in men than in women, whereas for DBP the relationship was linear for men only [38]. Notably, women constituted only 32% of the total sample.

Recommendations from the contemporary national and international guidelines advise to moderate alcohol intake to low-to-moderate alcohol consumption of up to 2 standard drinks per day for men and up to 1 drink per day for women in the context of managing hypertension. However, the safety and benefits of such low-to-moderate consumption are being called into question, as they may not be entirely risk-free.

Weighing the benefits of alcohol against the risks — is there somehow a right balance?

An important issue is addressing the total effect of alcohol and the controversies about the amount of alcohol associated with risk, which begs the question: is alcohol entirely bad?

While long-term heavy drinking is an established cause of hypertension, responsible for approximately 16% of cases of hypertension worldwide [39], findings from different studies on the effects of alcohol in CV disease (CVD) have been contradictory. Several epidemiological studies identified a protective association between low-to-moderate consumption of alcohol, BP, and CVD [40–42]. They reported a J-shaped or U-shaped association with benefits at low consumption and harmful effects at high consumption. The belief that alcohol could be beneficial for health is related to the "French paradox" based on findings from the WHO MONICA project [43]. This project, conducted primarily in Europe, aimed to explore the connection between saturated fat consumption and CVD mortality. A correlation between higher saturated fat intake and increased cardiovascular-related deaths was observed. However, this pattern did not hold true in all regions, with France presenting a striking anomaly. It exhibited notably high consumption of saturated fats but a lower than expected CV mortality rate. This intriguing phenomenon, labeled the "French paradox", was attributed to higher alcohol consumption in France, primarily in the form of red wine [43].

A meta-analysis of 10 studies [44] confirmed the J-shaped association between wine consumption and vascular events as well as CV mortality with a maximum protection observed at 21 g of wine per day [44]. Another comprehensive meta-analysis of one million participants found that low-to-moderate alcohol consumption is linked to significant reductions in total mortality [45]. In men, the maximum benefit was achieved at one to two drinks daily, leading to a 17% reduction in total mortality (95% CI, 15%–19%) and for women, half to one drink daily, resulting in an 18% decrease in total mortality (99% CI, 13%–22%). Intake levels exceeding 2.5 drinks per day for women and 4 drinks per day for men, were associated with progressively higher mortality rates in a dose-dependent manner [45].

In another study involving 245 000 individuals, both light (up to 3 drinks per week) and moderate drinkers (4–7 drinks per week for women, 4–14 drinks per week for men) drinking was linked to lower CV mortality when compared to heavy users (>7 drinks per week for women or >14 drinks per week for men) or those who abstained from alcohol [46].

Moreover, a pooled analysis of eight prospective studies encompassing 192 067 women and 74 919 men from North America and Europe found a negative association between alcohol intake and the risk of coronary artery disease [47].

A valid explanation to the "French paradox" and the seemingly protective behavior exhibited by wine as an alcoholic beverage at low consumption levels lies mainly in the presence of biological active compounds, namely, polyphenols, in its composition [48]. These include the flavonoids, e.g., quercetin, catechin, and the non-flavonoids, such as stilbenes (resveratrol and polydatin) [49]. The protective effect of the bioactive compounds in wine is attributed to their anti-oxidant, anti-thrombotic, and anti-inflammatory properties [50], which was confirmed by meta-analyses reporting on the beneficial effects of the polyphenols, from red wine or berries on the CV health [51, 52].

Furthermore, long term low-to-moderate wine consumption was reported to increased high-density lipoprotein levels, an effect mediated by ethanol, presumably by increasing hepatic lipoprotein production and transport rate [53]. However, certain factors challenge the robustness of the data substantiating this assertion [48]. The majority of these observational admit limitations in the study design allowing for residual confounding and bias. These include, for example, misclassification of former drinkers as nondrinkers. Former drinkers might have ceased alcohol consumption due to health-related factors such as serious illness thus overestimating the health benefits of alcohol in the other comparator group [54]. Many studies focus on cohorts with participants above 35 years of age, potentially biasing the assessment of lifetime drinking effects. Episodes of binge drinking are more common in one's 20s, and individuals who engaged in heavy or binge drinking during their 20s are more likely to become abstainers after the age of 35 [55]. As a result, their CV risk, even though they are categorized as nondrinkers in some studies, may be elevated due to the underestimation of heavy or binge drinking during their youth [56]. Inconsistency in participant self-reporting of the amount of alcohol consumed, and failure to estimate the actual consumption further add to the limitations. Moreover, individuals who consume modest amounts of alcohol tend to have higher socioeconomic status, adopt nutritious dietary habits, and engage in a more physically active way of life. These limitations highlight the complexity and challenges of studying the relationship between alcohol consumption and CV health due to many forms of bias introduced into the studies. Considering all these limitations, the CV benefits of low-to-moderate alcohol consumption remain questionable, or even overestimated.

How different levels of habitual alcohol consumption, from low to heavy, affect cardiovascular health?

The potential challenges associated with establishing causation through observational studies and the limited scope and duration of trials on moderate alcohol consumption have created a situation of uncertainty. While epidemiologic studies have advocated CV benefits with low alcohol intake, compared to either abstinence or heavy consumption, suggesting J- or U-shaped relationship, emerging evidence form Mendelian randomization (MR) studies suggests otherwise. MR studies use genetic information to mitigate confounding between exposure and outcome [57]. They can investigate causal connections by using naturally occurring genetic variations as impartial substitutes for an exposure [57], which is advantageous since genetic predisposition is unaffected by confounders. In this case, the residual confounding described earlier due to healthier lifestyle, socioeconomic, and behavioral factors that tend to coincide with modest alcohol drinking could be alleviated. A cohort study [58] using data from the UK Biobank (2006–2010, follow-up until 2016) involving 371 463 (54% women) individuals employed both linear and nonlinear MR in the analysis. The study demonstrated a consistent nonlinear, risk-increasing relationship between alcohol consumption and CV diseases (hypertension and coronary artery disease). The risk was modest at low consumption level but escalated at higher levels, indicating that even low alcohol intake was associated with a modest increase in risk. Therefore, it provided evidence that alcohol consumption, across all levels, is associated with increased CV risk including also the amounts considered harmless by current international guidelines. Moreover, the study reported attenuation in the apparent protective associations between modest alcohol intake and CV risk when confounding lifestyle factors were adjusted for [58]. Hence, the cardioprotective associations observed with the J- and U-shaped epidemiologic curves are largely unsubstantiated.

The idea of carrying out large multicenter randomized intervention trials to investigate the influence of moderate alcohol consumption on CV health appears promising but comes with significant challenges and complexities. In 2018, the National Institutes of Health in the United States initiated a randomized clinical trial (MACH 15) to examine the effects of daily moderate alcohol intake (11–15 g of ethanol or one standard drink) compared to abstention on the risk of major CV events [59]. However, this study was swiftly terminated due to ethical concerns and the potential for bias and conflict arising from industry influence [60].

Apart from the questionable CV benefits of low-to-moderate alcohol consumption, excessive drinking can lead to adverse health consequences. Heavy alcohol intake is dangerous and is a leading risk factor for death and disability. A study examining the impact of vodka consumption on premature death among 200 000 Russian adults found that heavy vodka consumption significantly increased the risk of death. Among male smokers who consumed ≥3 bottles of vodka per week, the 20-year risk of death was 35% and 64% at age range 35–54 and 55–74 years, respectively [61]. Another study [62] analyzed data from nearly 600 000 current drinkers in 19 high-income countries without prior CVD to determine the thresholds associated with the lowest risk of all-cause mortality and CVD. They found that, in both men and women, those who drink 100–≤200 g alcohol per week or higher have lower life expectancy at age 40 years compared to those who consume less. Moreover, alcohol consumption was linearly associated with higher CVD risk such as stroke, coronary disease, heart failure, of fatal hypertensive disease [62]. A threshold for the lowest risk of all-cause mortality of around 100 g of alcohol per week was suggested which is lower than the alcohol consumption limits recommended by most guidelines [62], highlighting the lack of a safe level.

Beyond hypertension: What other CV ramifications of alcohol consumptions should be considered?

There's a well-documented association between excessive alcohol consumption and an increased risk of numerous CV conditions, among which are arrhythmias, coronary artery disease, and cardiomyopathy [63]. These complications may arise due to prolonged hypertension or through various other pathophysiological mechanisms not directly related to hypertension. Excessive alcohol consumption significantly affects the heart, potentially triggering arrhythmias and, albeit infrequently, even leading to sudden cardiac death [64]. Hence, a term "holiday heart" was devised for acute arrhythmias, mostly atrial fibrillation (AF), in individuals who engage in heavy or binge drinking during festivities or celebrations [65]. Alcohol can induce arrhythmias through various mechanisms acting as a trigger for AF by disrupting the heart's electrical system, shortening the atrial effective refractory period and slowing atrial conduction [66]. It can disrupt electrolyte balance, impact the autonomic nervous system, directly affect cardiac cells, and increase the heart rate [66]. Habitual drinking contributes to a progressive remodeling of the atria and is a modifiable risk factor for AF and left atrial dilation [67]. A recent meta-analysis [68] analyzing data from 13 prospective studies encompassing over 10 million participants with 214 365 AF cases showed a 6% increase in AF risk per 1 drink per day (about 12 g) rise in alcohol intake across sexes (RR, 1.06; 95% CI, 1.03–1.08). The relationship was linear in men but J-shaped in women indicating that moderate alcohol consumption might have a different impact on AF risk in women [68]. Alcohol can also exacerbate the risk of developing AF by increasing BP while hypertension *per se* is a significant risk factor for developing AF [69]. Therefore, both hypertension and AF can be exacerbated or triggered by excessive alcohol consumption. Hence, alcohol consumption may potentially worsen the clinical presentation of hypertensive individuals and should be an integral part of comprehensive management of hypertension and arrhythmias to reduce the risk of complications.

While alcohol-induced BP elevation is a significant contributor to left ventricular dysfunction, the overall impact of alcohol on the heart is multifaceted. Alcohol exerts direct toxic effects on cardiac cells, propagates alcoholic cardiomyopathy and arrhythmias which are crucially involved in compromising the function of the left ventricle in individuals with alcohol abuse disorders [70]. Alcohol abuse exacerbates the complications of hypertension on the heart, particularly the left ventricle, significantly increasing the risk and severity of left ventricular dysfunction in individuals with both alcohol abuse and hypertension [71]. Chronic alcohol use can induce various echocardiographic alterations reflecting structural and functional damage caused by alcohol in the heart that vary based on the duration and extent of alcohol consumption. In a study, chronic alcohol intake over an average of 16 years in a dose of 4 alcohol units per day caused left ventricle hypertrophy, diastolic and systolic dysfunction with an increase in left atrial volume, decreased ejection fraction and impaired global and layer-specific longitudinal strain [72].

Finally, alcohol abuse can significantly contribute to the side effects observed in patients on antihypertensive medications by amplifying drug side effects including the risk of orthostatic hypotension and falls [73]. This is especially relevant for older adults who are more susceptible to these adverse effects. Antihypertensive drugs mostly implicated in this context are diuretics, vasodilators such alpha blockers, and centrally acting antihypertensives [74]. Alcohol exerts diuresis which, when combined with diuretics, might increase the risk of dehydration, exacerbating side effects such as dizziness or electrolyte imbalances. These interactions necessitate careful monitoring and clear guidance on alcohol use alongside medication to mitigate risks in older adults.

The prevalence of potentially serious alcohol-medication interactions in older adults was studied in the Irish Longitudinal Study on Ageing (TILDA) [75] cohort, which demonstrated an overall prevalence of potential interactions in 18% of participants with 8% at risk of one interaction and 10% at risk of at least two interactions. The most common interactions involved, indeed, CV agents with estimated 15% of older adults identified as being at risk of a serious alcohol-medication interaction.

A question to the physician: "Which lifestyle changes do you recommend to your patients with hypertension?"

Alcohol consumption should be assessed at the primary care level when dealing with elevated BP, which is advocated by the guidelines for hypertension management including the European guidelines. The latter specify that all patients should have their alcohol consumption assessed and encouraged to reduce their intake if they drink heavily [4]. However, it appears that addressing alcohol intake remains one of the least prioritized aspects of hypertension management [76].

A survey conducted among 1064 physicians assessed physician behavior regarding alcohol consumption screening and awareness of the European guidelines on moderate alcohol consumption [77]. The survey revealed that while 81.9% of physicians generally quantify alcohol consumption in hypertensive patients, only 28.6% screened alcohol consumption in patients with newly detected hypertension, and 14.5% in patients with treatment-resistant hypertension. The study highlighted a deficit in clinical practice and the need for improved screening and management of alcohol consumption in hypertensive patients [77].

How effective is alcohol reduction in lowering BP? Is there a threshold for the effect?

A systematic review and meta-analysis [78] analyzed 36 randomized controlled trials involving 2865 participants (of whom 401 were women) and showed that moderating

alcohol consumption has the potential to lower BP in a dose-dependent manner, suggesting a threshold effect of ≥3 drinks per day. The study found that the most substantial reduction in SBP (–5.5 mm Hg [95% CI, –6.7 to –4.3]) and DBP (–4.0 mm Hg [95% CI, –4.7 to –3.3]) was among individuals consuming ≥6 drinks per day when their alcohol intake was reduced by 50%. The results may be more applicable to men, as women constituted only a small fraction of the participants in the trials included.

CONCLUSION

Moderation in alcohol intake and implementation of alcohol-free days during the week in both men and women who consume drinks that contain alcohol represents an important lifestyle intervention in patients with hypertension and is recommended to improve BP control and overall health.

Article information

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REFERENCES

1. Total per capita (15+) consumption (in litres of pure alcohol) by WHO region. https://apps.who.int/gho/data/view.gisah.A1029SDG3REGv?lang=en&showonly=GISAH (accessed: September 19, 2023).
2. GBD 2019 Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020; 396(10258): 1223–1249, doi: 10.1016/S0140-6736(20)30752-2, indexed in Pubmed: 33069327.
3. Mongan D, Long J. Standard drink measures throughout Europe; peoples' understanding of standard drinks. RARHA: Joint Actional on Reducing Alcohol Related Harm 2015.
4. Mancia G, Kreutz R, Brunström M, et al. 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens. 2023; 41(12): 1874–2071, doi: 10.1097/HJH.0000000000003480, indexed in Pubmed: 37345492.
5. Kalinowski A, Humphreys K. Governmental standard drink definitions and low-risk alcohol consumption guidelines in 37 countries. Addiction. 2016; 111(7): 1293–1298, doi: 10.1111/add.13341, indexed in Pubmed: 27073140.
6. Babor T, Higgins-Biddle J, Saunders J, et al. AUDIT the alcohol user disorders identification test second edition. Genebra: World Health Organization, 2001.
7. Charchar FJ, Prestes PR, Mills C, et al. Lifestyle management of hypertension: International Society of Hypertension position paper endorsed by the World Hypertension League and European Society of Hypertension. J Hypertens. 2024; 42(1): 23–49, doi: 10.1097/HJH.0000000000003563, indexed in Pubmed: 37712135.
8. Frezza M, di Padova C, Pozzato G, et al. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. N Engl J Med. 1990; 322(2): 95–99, doi: 10.1056/NEJM199001113220205, indexed in Pubmed: 2248624.
9. Seppä K, Laippala P, Sillanaukee P, et al. Drinking pattern and blood pressure. Am J Hypertens. 1994; 7(3): 249–254, doi: 10.1093/ajh/7.3.249, indexed in Pubmed: 8003276.
10. Williams B, Mancia G, Spiering W, et al. 2018 Practice Guidelines for the management of arterial hypertension of the European Society of Hypertension and the European Society of Cardiology: ESH/ESC Task Force for the Management of Arterial Hypertension. J Hypertens. 2018; 36(12): 2284–2309, doi: 10.1097/HJH.0000000000001961, indexed in Pubmed: 30379783.
11. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. Hypertension. 2018; 71(6): e13–e115, doi: 10.1161/HYP.0000000000000065, indexed in Pubmed: 29133356.
12. Unger T, Borghi C, Charchar F, et al. 2020 International Society of Hypertension Global Hypertension Practice Guidelines. Hypertension. 2020; 75(6): 1334–1357, doi: 10.1161/HYPERTENSIONAHA.120.15026, indexed in Pubmed: 32370572.
13. Tykarski A, Filipiak K, Januszewicz A, et al. 2019 Guidelines for the management of hypertension — part 1–7. Arterial Hypertension. 2019; 23(2): 41–87, doi: 10.5603/ah.a2019.0008.
14. Joint Committee for Guideline Revision. 2018 Chinese guidelines for prevention and treatment of hypertension — a report of the Revision Committee of Chinese Guidelines for Prevention and Treatment of Hypertension. J Geriatr Cardiol. 2019; 16(3): 182–241, doi: 10.11909/j.issn.1671-5411.2019.03.014, indexed in Pubmed: 31080465.
15. Lee HY, Shin J, Kim GH, et al. 2018 Korean Society of Hypertension Guidelines for the management of hypertension: part II-diagnosis and treatment of hypertension. Clin Hypertens. 2019; 25: 20, doi: 10.1186/s40885-019-0124-x, indexed in Pubmed: 31388453.
16. Umemura S, Arima H, Arima S, et al. The Japanese Society of Hypertension guidelines for the management of hypertension (JSH 2019). Hypertens Res. 2019; 42(9): 1235–1481, doi: 10.1038/s41440-019-0284-9, indexed in Pubmed: 31375757.
17. Husain K, Ansari RA, Ferder L. Alcohol-induced hypertension: Mechanism and prevention. World J Cardiol. 2014; 6(5): 245–252, doi: 10.4330/wjc.v6.i5.245, indexed in Pubmed: 24891935.
18. Puddey IB, Zilkens RR, Croft KD, et al. Alcohol and endothelial function: a brief review. Clin Exp Pharmacol Physiol. 2001; 28(12): 1020–1024, doi: 10.1046/j.1440-1681.2001.03572.x, indexed in Pubmed: 11903307.
19. Fuchs FD, Chambless LE, Whelton PK, et al. Alcohol consumption and the incidence of hypertension: The Atherosclerosis Risk in Communities Study. Hypertension. 2001; 37(5): 1242–1250, doi: 10.1161/01.hyp.37.5.1242, indexed in Pubmed: 11358935.
20. Tasnim S, Tang C, Musini VM, et al. Effect of alcohol on blood pressure. Cochrane Database Syst Rev. 2020; 7(7): CD012787, doi: 10.1002/14651858.CD012787.pub2, indexed in Pubmed: 32609894.
21. Piano MR, Mazzuco A, Kang M, et al. Cardiovascular consequences of binge drinking: An integrative review with implications for advocacy, policy, and research. Alcohol Clin Exp Res. 2017; 41(3): 487–496, doi: 10.1111/acer.13329, indexed in Pubmed: 28067964.
22. Stranges S, Wu T, Dorn JM, et al. Relationship of alcohol drinking pattern to risk of hypertension: a population-based study. Hypertension. 2004; 44(6): 813–819, doi: 10.1161/01.HYP.0000146537.03103.f2, indexed in Pubmed: 15477381.
23. Kang M, Phillips SA, Piano MR. Relationship between cardiovascular risk factors and binge drinking among college students in South Korea. J Ethn Subst Abuse. 2020; 19(1): 119–132, doi: 10.1080/15332640.2018.1484311, indexed in Pubmed: 30064300.
24. Piano MR, Burke L, Kang M, et al. Effects of repeated binge drinking on blood pressure levels and other cardiovascular health metrics in young adults: national health and nutrition examination survey, 2011–2014. J Am Heart Assoc. 2018; 7(13), doi: 10.1161/JAHA.118.008733, indexed in Pubmed: 29950486.
25. Hayibor LA, Zhang J, Duncan A. Association of binge drinking in adolescence and early adulthood with high blood pressure: findings from the National Longitudinal Study of Adolescent to Adult Health (1994–2008). J Epidemiol Community Health. 2019; 73(7): 652–659, doi: 10.1136/jech-2018-211594, indexed in Pubmed: 30971421.
26. Laatikainen T, Manninen L, Poikolainen K, et al. Increased mortality related to heavy alcohol intake pattern. J Epidemiol Community Health. 2003; 57(5): 379–384, doi: 10.1136/jech.57.5.379, indexed in Pubmed: 12700224.

27. Sundell L, Salomaa V, Vartiainen E, et al. Increased stroke risk is related to a binge-drinking habit. Stroke. 2008; 39(12): 3179–3184, doi: 10.1161/STROKEAHA.108.520817, indexed in Pubmed: 18832741.
28. Seppä K, Sillanaukee P. Binge drinking and ambulatory blood pressure. Hypertension. 1999; 33(1): 79–82, doi: doi:10.1161/01.HYP.33.1.79, indexed in Pubmed: 9931085..
29. Peng J, Wang H, Rong X, et al. Cerebral hemorrhage and alcohol exposure: A review. Alcohol Alcohol. 2020; 55(1): 20–27, doi: 10.1093/alcalc/agz087, indexed in Pubmed: 31845978.
30. Ding J, Sigurdsson S, Garcia M, et al. Risk factors associated with incident cerebral microbleeds according to location in older people: The age, gene/environment susceptibility (ages) — Reykjavik study. JAMA Neurol. 2015; 72(6): 682–688, doi: 10.1001/jamaneurol.2015.0174, indexed in Pubmed: 25867544.
31. Ariesen MJ, Claus SP, Rinkel GJE, et al. Risk factors for intracerebral hemorrhage in the general population: A systematic review. Stroke. 2003; 34(8): 2060–2065, doi: 10.1161/01.STR.0000080678.09344.8D, indexed in Pubmed: 12843354.
32. Puy L, Parry-Jones AR, Sandset EC, et al. Intracerebral haemorrhage. Nat Rev Dis Primers. 2023; 9(1): 14, doi: 10.1038/s41572-023-00424-7, indexed in Pubmed: 36928219.
33. Schoenborn CA, Stommel M, Ward BW. Mortality risks associated with average drinking level and episodic heavy drinking. Subst Use Misuse. 2014; 49(10): 1250–1258, doi: 10.3109/10826084.2014.891620, indexed in Pubmed: 24621084.
34. Lian C. Alcoholism: cause of arterial hypertension. Bull Acad Med. 1915; 74: 525–528.
35. Fuchs FD, Fuchs SC. The effect of alcohol on blood pressure and hypertension. Curr Hypertens Rep. 2021; 23(10): 42, doi: 10.1007/s11906-021-01160-7, indexed in Pubmed: 34762198.
36. Roerecke M, Tobe SW, Kaczorowski J, et al. Sex-specific associations between alcohol consumption and incidence of hypertension: A systematic review and meta-analysis of cohort studies. J Am Heart Assoc. 2018; 7(13): e008202, doi: 10.1161/JAHA.117.008202, indexed in Pubmed: 29950485.
37. Liu F, Liu Y, Sun X, et al. Race- and sex-specific association between alcohol consumption and hypertension in 22 cohort studies: A systematic review and meta-analysis. Nutr Metab Cardiovasc Dis. 2020; 30(8): 1249–1259, doi: 10.1016/j.numecd.2020.03.018, indexed in Pubmed: 32446870.
38. Di Federico S, Filippini T, Whelton PK, et al. Alcohol intake and blood pressure levels: A dose-response meta-analysis of nonexperimental cohort studies. Hypertension. 2023; 80(10): 1961–1969, doi: 10.1161/HYPERTENSIONAHA.123.21224, indexed in Pubmed: 37522179.
39. Puddey IB, Beilin LJ. Alcohol is bad for blood pressure. Clin Exp Pharmacol Physiol. 2006; 33(9): 847–852, doi: 10.1111/j.1440-1681.2006.04452.x, indexed in Pubmed: 16922819.
40. Grønbaek M. Alcohol, type of alcohol, and all-cause and coronary heart disease mortality. Ann N Y Acad Sci. 2002; 957: 16–20, doi: 10.1111/j.1749-6632.2002.tb02902.x, indexed in Pubmed: 12074958.
41. Bell S, Daskalopoulou M, Rapsomaniki E, et al. Association between clinically recorded alcohol consumption and initial presentation of 12 cardiovascular diseases: population based cohort study using linked health records. BMJ. 2017; 356: j909, doi: 10.1136/bmj.j909, indexed in Pubmed: 28331015.
42. Yu A, Cooke AB, Scheffler P, et al. Alcohol exerts a shifted U-shaped effect on central blood pressure in young adults. J Gen Intern Med. 2021; 36(10): 2975–2981, doi: 10.1007/s11606-021-06665-0, indexed in Pubmed: 33674917.
43. Ferrières J. The French paradox: lessons for other countries. Heart. 2004; 90(1): 107–111, doi: 10.1136/heart.90.1.107, indexed in Pubmed: 14676260.
44. Costanzo S, Di Castelnuovo A, Donati MB, et al. Wine, beer or spirit drinking in relation to fatal and non-fatal cardiovascular events: a meta-analysis. Eur J Epidemiol. 2011; 26(11): 833–850, doi: 10.1007/s10654-011-9631-0, indexed in Pubmed: 22076059.
45. Di Castelnuovo A, Costanzo S, Bagnardi V, et al. Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. Arch Intern Med. 2006; 166(22): 2437–2445, doi: 10.1001/archinte.166.22.2437, indexed in Pubmed: 17159008.
46. Mukamal KJ, Chen CM, Rao SR, et al. Alcohol consumption and cardiovascular mortality among U.S. adults, 1987 to 2002. J Am Coll Cardiol. 2010; 55(13): 1328–1335, doi: 10.1016/j.jacc.2009.10.056, indexed in Pubmed: 20338493.
47. Hvidtfeldt UA, Tolstrup JS, Jakobsen MU, et al. Alcohol intake and risk of coronary heart disease in younger, middle-aged, and older adults. Circulation. 2010; 121(14): 1589–1597, doi: 10.1161/CIRCULATIONAHA.109.887513, indexed in Pubmed: 20351238.
48. Chiva-Blanch G, Badimon L. Benefits and risks of moderate alcohol consumption on cardiovascular disease: current findings and controversies. Nutrients. 2019; 12(1): 108, doi: 10.3390/nu12010108, indexed in Pubmed: 31906033.
49. Waterhouse AL. Wine phenolics. Ann N Y Acad Sci. 2002; 957(1): 21–36, doi: 10.1111/j.1749-6632.2002.tb02903.x, indexed in Pubmed: 12074959.
50. Fragopoulou E, Antonopoulou S. The French paradox three decades later: Role of inflammation and thrombosis. Clin Chim Acta. 2020; 510: 160–169, doi: 10.1016/j.cca.2020.07.013, indexed in Pubmed: 32653485.
51. Weaver SR, Rendeiro C, McGettrick HM, et al. Fine wine or sour grapes? A systematic review and meta-analysis of the impact of red wine polyphenols on vascular health. Eur J Nutr. 2021; 60(1): 1–28, doi: 10.1007/s00394-020-02247-8, indexed in Pubmed: 32303823.
52. Huang H, Chen G, Liao D, et al. Effects of berries consumption on cardiovascular risk factors: A meta-analysis with trial sequential analysis of randomized controlled trials. Sci Rep. 2016; 6: 23625, doi: 10.1038/srep23625, indexed in Pubmed: 27006201.
53. Fragopoulou E, Choleva M, Antonopoulou S, et al. Wine and its metabolic effects. A comprehensive review of clinical trials. Metabolism. 2018; 83: 102–119, doi: 10.1016/j.metabol.2018.01.024, indexed in Pubmed: 29408458.
54. Tsubono Y, Yamada S, Nishino Y, et al. Choice of comparison group in assessing the health effects of moderate alcohol consumption. JAMA. 2001; 286(10): 1177–1178, doi: 10.1001/jama.286.10.1177, indexed in Pubmed: 11559261.
55. Britton A, Ben-Shlomo Y, Benzeval M, et al. Life course trajectories of alcohol consumption in the United Kingdom using longitudinal data from nine cohort studies. BMC Med. 2015; 13: 47, doi: 10.1186/s12916-015-0273-z, indexed in Pubmed: 25858476.
56. Russell M, Fan AZ, Freudenheim JoL, et al. Lifetime drinking trajectories and nonfatal acute myocardial infarction. Alcohol Clin Exp Res. 2019; 43(11): 2384–2394, doi: 10.1111/acer.14190, indexed in Pubmed: 31566766.
57. Smith GD, Ebrahim S. 'Mendelian randomization': can genetic epidemiology contribute to understanding environmental determinants of disease? Int J Epidemiol. 2003; 32(1): 1–22, doi: 10.1093/ije/dyg070, indexed in Pubmed: 12689998.
58. Biddinger KJ, Emdin CA, Haas ME, et al. Association of habitual alcohol intake with risk of cardiovascular disease. JAMA Netw Open. 2022; 5(3): e223849, doi: 10.1001/jamanetworkopen.2022.3849, indexed in Pubmed: 35333364.
59. Spiegelman D, Lovato LC, Khudyakov P, et al. The Moderate Alcohol and Cardiovascular Health Trial (MACH15): Design and methods for a randomized trial of moderate alcohol consumption and cardiometabolic risk. Eur J Prev Cardiol. 2020; 27(18): 1967–1982, doi: 10.1177/2047487320912376, indexed in Pubmed: 32250171.
60. Babor TF. Big alcohol meets big science at NIAAA: what could go wrong? J Stud Alcohol Drugs. 2023; 84(1): 5–10, doi: 10.15288/jsad.22-00434, indexed in Pubmed: 36799669.
61. Zaridze D, Lewington S, Boroda A, et al. Alcohol and mortality in Russia: prospective observational study of 151,000 adults. Lancet. 2014; 383(9927): 1465–1473, doi: 10.1016/S0140-6736(13)62247-3, indexed in Pubmed: 24486187.
62. Wood AM, Kaptoge S, Butterworth AS, et al. Risk thresholds for alcohol consumption: combined analysis of individual-participant data for 599 912 current drinkers in 83 prospective studies. Lancet. 2018; 391(10129): 1513–1523, doi: 10.1016/S0140-6736(18)30134-X, indexed in Pubmed: 29676281.
63. Piano MR. Alcohol's effects on the cardiovascular system. Alcohol Res. 2017; 38(2): 219–241, indexed in Pubmed: 28988575.
64. Albert CM, Manson JE, Cook NR, et al. Moderate alcohol consumption and the risk of sudden cardiac death among US male physicians. Circulation. 1999; 100(9): 944–950, doi: 10.1161/01.cir.100.9.944, indexed in Pubmed: 10468525.

65. Ettinger PO, Wu CF, De La Cruz C, et al. Arrhythmias and the "Holiday Heart": alcohol-associated cardiac rhythm disorders. Am Heart J. 1978; 95(5): 555–562, doi: 10.1016/0002-8703(78)90296-x, indexed in Pubmed: 636996.
66. Fernández-Solà J. Cardiovascular risks and benefits of moderate and heavy alcohol consumption. Nat Rev Cardiol. 2015; 12(10): 576–587, doi: 10.1038/nrcardio.2015.91, indexed in Pubmed: 26099843.
67. Voskoboinik A, Wong G, Lee G, et al. Moderate alcohol consumption is associated with atrial electrical and structural changes: Insights from high-density left atrial electroanatomic mapping. Heart Rhythm. 2019; 16(2): 251–259, doi: 10.1016/j.hrthm.2018.10.041, indexed in Pubmed: 30639070.
68. Jiang H, Mei X, Jiang Y, et al. Alcohol consumption and atrial fibrillation risk: An updated dose-response meta-analysis of over 10 million participants. Front Cardiovasc Med. 2022; 9: 979982, doi: 10.3389/fcvm.2022.979982, indexed in Pubmed: 36247447.
69. Kallistratos MS, Poulimenos LE, Manolis AJ. Atrial fibrillation and arterial hypertension. Pharmacol Res. 2018; 128: 322–326, doi: 10.1016/j.phrs.2017.10.007, indexed in Pubmed: 29055746.
70. Gonçalves A, Jhund PS, Claggett B, et al. Relationship between alcohol consumption and cardiac structure and function in the elderly: the Atherosclerosis Risk In Communities Study. Circ Cardiovasc Imaging. 2015; 8(6), doi: 10.1161/CIRCIMAGING.114.002846, indexed in Pubmed: 26015266.
71. Fernández-Solà J, Nicolás JM, Paré JC, et al. Diastolic function impairment in alcoholics. Alcohol Clin Exp Res. 2000; 24(12): 1830–1835, indexed in Pubmed: 11141042.
72. Hamala P, Kasprzak JD, Bińkowska A, et al. The impact of chronic alcohol overuse on heart function and prognosis: layer-specific longitudinal strain and mid-term outcome analysis. Kardiol Pol. 2021; 79(7-8): 781–788, doi: 10.33963/KP.15986, indexed in Pubmed: 33926169.
73. Narkiewicz K, Cooley RL, Somers VK. Alcohol potentiates orthostatic hypotension : implications for alcohol-related syncope. Circulation. 2000; 101(4): 398–402, doi: 10.1161/01.cir.101.4.398, indexed in Pubmed: 10653831.
74. Moore AA, Whiteman EJ, Ward KT. Risks of combined alcohol/medication use in older adults. Am J Geriatr Pharmacother. 2007; 5(1): 64–74, doi: 10.1016/j.amjopharm.2007.03.006, indexed in Pubmed: 17608249.
75. Holton A, Boland F, Gallagher P, et al. Longitudinal prevalence of potentially serious alcohol-medication interactions in community-dwelling older adults: a prospective cohort study. Eur J Clin Pharmacol. 2019; 75(4): 569–575, doi: 10.1007/s00228-018-02608-7, indexed in Pubmed: 30569283.
76. Rehm J, Prieto JA, Beier M, et al. The role of alcohol in the management of hypertension in patients in European primary health care practices - a survey in the largest European Union countries. BMC Fam Pract. 2016; 17(1): 130, doi: 10.1186/s12875-016-0529-5, indexed in Pubmed: 27608770.
77. Zaidi Touis L, Bolbrinker J, Riemer TG, et al. Moderation of alcohol consumption as a recommendation in European hypertension management guidelines: a survey on awareness, screening and implementation among European physicians. BMJ Open. 2018; 8(10): e022026, doi: 10.1136/bmjopen-2018-022026, indexed in Pubmed: 30344170.
78. Roerecke M, Kaczorowski J, Tobe SW, et al. The effect of a reduction in alcohol consumption on blood pressure: a systematic review and meta-analysis. Lancet Public Health. 2017; 2(2): e108–e120, doi: 10.1016/S2468-2667(17)30003-8, indexed in Pubmed: 29253389.